Point-of-Care Echocardiography Improves Assessment of Volume Status in Cirrhosis and Hepatorenal Syndrome

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ABSTRACT

The management of patients with cirrhosis along with acute kidney injury is complex and depends in large part on accurate assessment of intravascular volume status. Assessment of intravascular volume status by point-of-care echocardiography often relies solely on inferior vena cava size and variability evaluation; however, this parameter should be interpretated with an understanding of right ventricular function integrated with stroke volume and flow. Attempts to optimize intra-abdominal hemodynamics favorably are clearly problematic when physical examination findings or rudimentary assessments of central venous pressure or change in central venous pressure are used. Here, we have demonstrated the potential utility of point-of-care echocardiography to optimize the hemodynamic state in patients with decompensated cirrhosis along with acute kidney injury. This case is very unique and describes how this technique may have great promise in optimizing the intra-abdominal hemodynamics and predict the timing of large-volume paracentesis in patients with decompensated cirrhosis, which in turn can aid in promoting favorable renal recovery.

Key Indexing Terms: Acute kidney injury; Hepatorenal syndrome; Liver failure; Ascites; Point-of-care echocardiography.
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CASE PRESENTATION

A 66-year-old woman with hepatitis C virus–related cirrhosis, portal hypertension, ascites, esophageal varices and chronic kidney disease (baseline creatinine of 1.7 mg/dL) presented with acute kidney injury (AKI) and altered mental status. She was believed to have hepatic encephalopathy and required intubation for airway protection on hospital day 2. Her serum creatinine level was 3.1 mg/dL at presentation. To manage her AKI, she received an aggressive volume-loading strategy with both crystalloid and colloid, and a 4.8-Liter paracentesis was performed. A total of 13.3 L of crystalloid plus 100 g of albumin was given with a total urine output (UOP) of 6.6 L over 12 days (yielding a positive fluid balance in excess of 7 L). Her daily average UOP was approximately 550 mL/day.

Owing to concerns about her elevated creatinine and poor UOP, the diagnosis of hepatorenal syndrome (HRS) was considered and a Nephrology consultation was obtained. A renal ultrasound was normal, urine sodium was <10 meq/L and the urinalysis was unremarkable. She received norepinephrine to augment mean arterial pressure (baseline mean arterial pressures were 60-65 mm Hg, with the goal to be 80 mm Hg or greater) with ongoing fluid administration. After receiving 4 days of norepinephrine (dose range of 2-8 mcg/kg/minute) and upon transfer to the ward, she was transitioned to midodrine and octreotide. Her serum creatinine level improved from 2.7-2.2 mg/dL and her UOP ranged from 490-615 mL while on norepinephrine. Following extubation, her central venous pressure (CVP) was 8-9 mm Hg and examination was notable for an edematous state.

Over the next 2 days, she developed worsening oliguria with a UOP totaling of 100 cc/day. Point-of-care echocardiography (POCE) was performed to gain a better understanding of her intravascular volume status because of a lack of response to fluid and concerns of underlying HRS.

The POCE revealed that her initial inferior vena cava (IVC) measured 0.44 cm in diameter with minimal respiratory variation (Figure 1A). Pulse-wave Doppler from an apical 5-chamber view of the left ventricular outflow tract (LVOT) showed a velocity time integral (VTI) increase from 25.7 cm (Figure 1B) to 34.8 cm (Figure 1C) following a passive leg-raise maneuver, consistent with an elevated stroke volume and cardiac output at baseline. In Figure 1D and E, the pulse-wave Doppler of the mitral inflow from an apical 4-chamber view is shown. In Figure 1D, pre-passive leg raise reveals an *E* velocity of 73.2 cm/second, with *E/A* reversal. In Figure 1E, the *E* velocity increased to 97.6 cm/second with normalization of the *E/A* pattern corresponding to a 33% increase in preload augmentation. This mitral inflow pattern with corresponding change with passive leg raise combined with an IVC maximal diameter of <1 cm is consistent with hypovolemia. Based on these findings, she received albumin at 25-g intravenous quarterly 6 hours for 1 day and was reassessed.

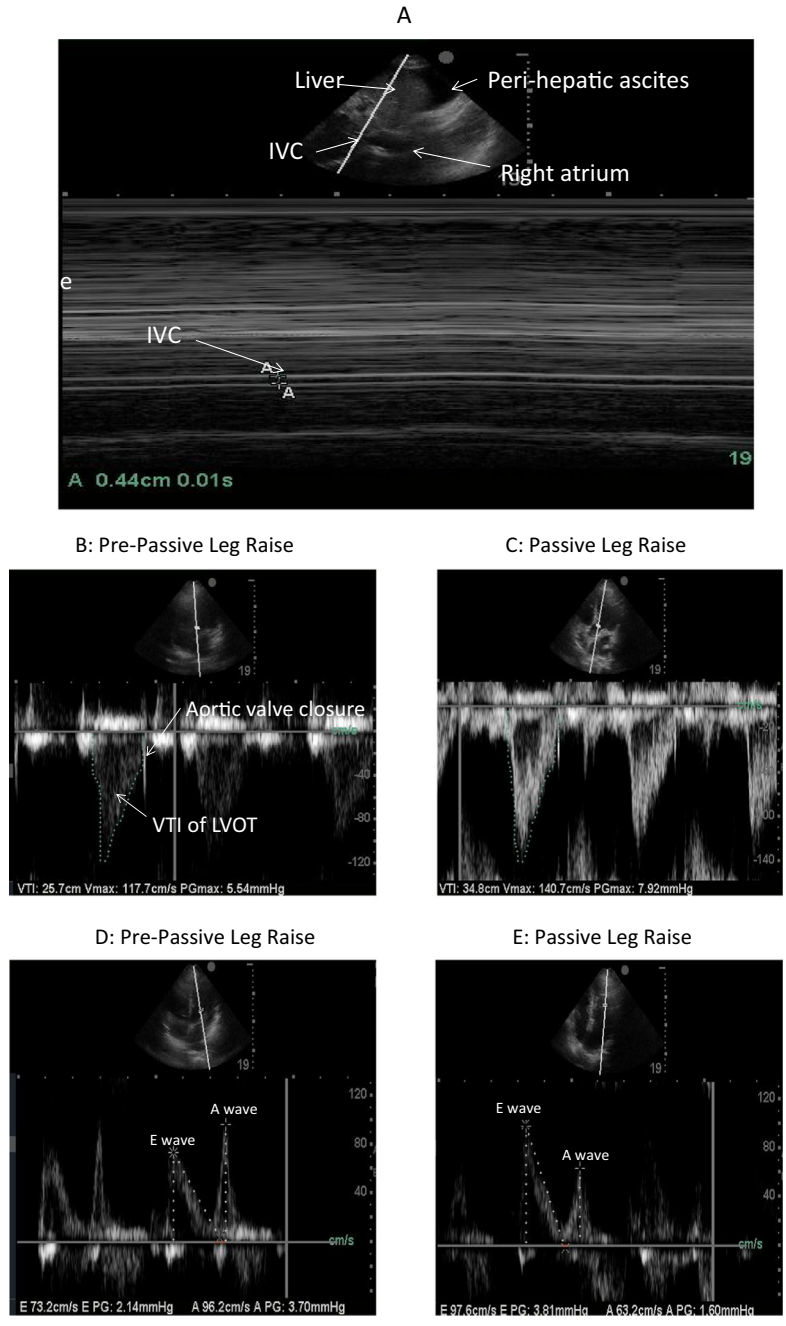

FIGURE 1. POCE evaluation of volume status preparacentesis. A, A 2-D and M-mode image of the IVC obtained from a subcostal view on a longitudinal axis is shown. The IVC measures 0.44 cm with minimal respiratory variation. This IVC size is indicative of a state of hypovolemia; however, the presence of significant intra-abdominal hypertension because of the presence of ascites may render a similar appearance. B and C, Pulse-wave Doppler images from an apical 5-chamber view of the left ventricular outflow tract (LVOT) showing a velocity time integral (VTI) increase from 25.7 cm (B) to 34.8 cm (C) following a passive leg-raise maneuver. This corresponds to a 35% increase in stroke volume. The area under the curve (designated by the white dots surrounding the Doppler wave (on the left side of each image) corresponds to the VTI of the LVOT and when multiplied by the cross surface area of the LVOT yields stroke volume in milliliters. To calculate stroke volume, the VTI (in cm) is multiplied by the cross surface area of the LVOT (in cm^2). D and E, Pulse-wave Doppler of mitral inflow from an apical 4-chamber view is shown. D, Pre-passive leg raise reveals an *E* velocity of 73.2 cm/second, with *E/A* reversal. E, The *E* velocity increased to 97.6 cm/second with normalization of the *E/A* pattern corresponding to a 33% increase in preload augmentation. This mitral inflow pattern with corresponding change with passive leg raise is consistent with hypovolemia. 2-D, 2-dimensional; IVC, inferior vena cava; POCE, point-of-care echocardiography; LVOT, left ventricular outflow tract; VTI, velocity time integral.